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Physiological Effects of Supplemental Concentrated Oxygen in VO_2 Kinetics During Steady-State Exercise

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Abstract

The administration of concentrated oxygen during exercise has continuously been questioned on its affiliation with increased athletic performance and recovery rates. To assess the validity of these statements, three untrained individuals performed two separate trials of steady-state exercise for 15 minutes in a cohort study. During the independent trial, a 2-liter pocket sized Boost Oxygen canister supplement containing 95% concentrated O_2 was self-administered by the subjects commencing their exercise. The study's findings suggest that concentrated O_2 supplements can increase VO_2 kinetics, resulting in a decreased time to attain steady-state VO_2. Furthermore, supplemental O_2 may help maintain a steadier steady-state VO_2 during aerobic exercise.

Keywords: Aerobic exercise, maximum oxygen uptake, oxygen deficit, trained athletes, untrained athletes

Introduction

For many years, athletes, trainers, and active individuals alike have been finding new ways to manipulate oxygen for improvement in performance and recovery rates. Techniques such as altitude masks, altitude training, and hyperoxic training have all been used to seek an advantage in competition and/or personal performance. Of these techniques, a popular hyperoxic method amongst athletes and active individuals is the administration of supplemental concentrated oxygen following exercise, due to its benefits in aiding with recovery. Hyperbaric oxygen therapy allows athletes to utilize oxygen for its claimed benefits to facilitate a faster recovery time resulting in fewer injuries (Babul and Rhodes, 2000). In R. G. Bannister and D. J. Cunningham's 1953 study, they tested the effects of concentrated oxygen on athletes and nonathletes at varied levels during treadmill runs. Their findings suggested that *any* addition of pure oxygen to inspired air "always improved performance considerably".

Although the use of concentrated oxygen had previously been banned in competition, as of 2010 the World Anti-Doping Agency (WADA) indicated that it "no longer prohibits supplemental oxygen (hyperoxia)". Since then, many athletes have sought out the benefits of using supplemental oxygen. The National Football League (NFL) has also allowed teams to supply concentrated oxygen for their players to use during football games for its supposed benefits. The NFL athletes can be seen on

the sidelines during games wearing oxygen masks, self-administering concentrated O_2 while they are momentarily off the field.

In this study, alternatively to studying the effects of concentrated oxygen during workouts, in-between workouts, or after workouts, we were interested in the physiological effects of supplemental concentrated O_2 intake as it pertains to VO_2 kinetics and oxygen deficit (otherwise known as oxygen debt) at the initial stages of a steady-state aerobic exercise. VO_2 kinetics refers to the time it takes for the body to increase mitochondrial enzyme production of ATP from the use of inspired oxygen to produce the energy that is needed for the exercise being performed. The initial debt of required oxygen required for the exercise is oxygen deficit. Oxygen deficit is the difference between oxygen uptake of the body during early stages of exercise and during a similar duration in a steady-state of exercise.

For trained individuals, their energy pathways take less time to reach steady-state VO_2 in comparison to an untrained individual. This difference can be represented in figures 1 & 2. According to the American College of Sports Medicine (ACSM) guidelines, an aerobically trained individual is someone who aerobically exercises at moderate intensity for 150 minutes per week. Since there is a deficit of oxygen at the beginning of workouts, we were curious to see if supplemental concentrated oxygen would have a positive effect on VO_2, O_2 deficit and reduce the time an individual's body takes to reach steady-state.

In a San Diego University physiological laboratory, three aerobically untrained male subjects were measured and tested in a cohort study. Testing the effects of Boost Oxygen's supplemental concentrated oxygen canister, containing 95% pure oxygen, in an aerobic exercise trial where the subjects were to maintain 70% of their heart rate maximum at a steady-state using a treadmill run and analyzed the data as it pertains to oxygen deficit and oxygen kinetics. We hypothesized that if the subjects administered the concentrated oxygen immediately after their warm-up but before steady-state VO_2, there will be an acceleration in VO_2 kinetics, a decrease in O_2 deficit, and provide faster cool-down rates during excess post-exercise oxygen consumption (EPOC) .

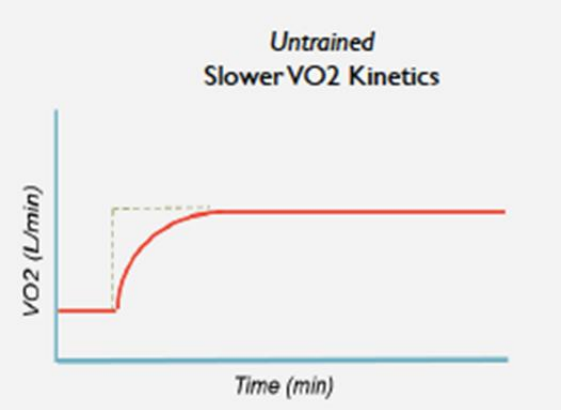

Figure 1. This graph is representative of the time for an untrained athlete to reach steady-state VO_2. The untrained individual's energy pathway systems take an extended time to reach steady-state VO_2.

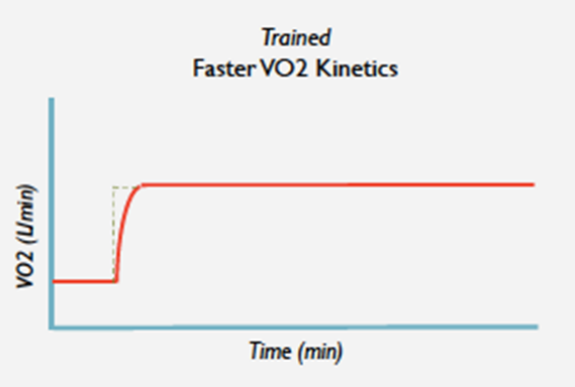

Figure 2. This graph is representative of the time for a trained athlete to reach steady-state VO_2. In comparison to the untrained subject, the time is decreased. This is due to the trained individual's energy pathway systems ability to reach steady-state VO_2 quicker.

Methods and Materials

The cohort study was performed in a university physiological laboratory. The laboratory's Parvo Medics' TrueOne® 2400 spirometer was the metabolic measurement system used for this experiment. The laboratory also had specialized treadmills which were used for the exercise trials. Three aerobically untrained male subjects were measured and tested. The subjects' measurables averaged to a mean weight of 77.4±14.1 kg, a mean height of 173±5 cm, and a mean age of 23±3 years. All the subjects were young, healthy, active, but did not meet the ACSM guideline requirement to be considered a trained aerobic athlete. The subjects all lived in the San Diego area and were attending the university during the trials.

The TrueOne spirometer was calibrated prior to commencing each of the subject's trials. Throughout the exercise trial, the subjects wore a breathing mask from the TrueOne spirometer that allowed for the subject's expired CO_2 to be measured and recorded. The variables that were measured and recorded were rating of perceived exertion (RPE), respiratory exchange ratio (RER), heart rate (HR), VO_2 relative and VO_2 absolute. For the recording of RPE, the subjects gave the test taker a number between 1-10 on their perception of how much exertion their workout was at that moment in time. The RPE was recorded every two minutes throughout the trial. The heart rate was measured a fingertip oximeter. The HR was also recorded every two minutes. The calculated target heart rate was found by using the first equation in figure 3 and multiplying it by (0.7). The RER, VO_2 relative and VO_2 absolute values were given by the TrueOne spirometer and recorded every two minutes.

The trials consisted of a two-minute warm-up, fifteen minutes of steady jogging/running exercise on a treadmill at a pace that was equivalent to the subject's 70% HR_{max} and a two-minute cool-down. The warm-up consisted of a moderate walk with an RPE of no more than 1. The subjects performed a control trial first. In the control trial, no supplemental oxygen was given. After one week, the subjects performed their second, independent trial with the same procedures as the first trial but with the introduction of supplemental concentrated oxygen. In the second trial the subjects were given a new and sealed 2-liter pocket sized Boost Oxygen canister containing 95% concentrated oxygen. Commencing their second trial, the subjects began the warm-up exactly like the first trial. Immediately following their warm-up, the subjects self-administered five deep inhalations of the oxygen canister, while exhaling back into the spirometer face mask after each inhalation of the supplemental oxygen. The subjects attempted to maintain their run during inhalations. Once the subjects finished their five inhalations, they then continued their fifteen-minute exercise at 70% HR_{max}, then proceeded to perform a two-minute cool-down. Concluding the trials, the results from the TrueOne spirometer were recorded.

TABLE 6.2
Commonly Used Equations for Estimating Maximal Heart Rate

Author	Equation	Population
Fox et al. (35)	HR_{max} = 220 − age	Small group of men and women
Astrand (8)	HR_{max} = 216.6 − (0.84 × age)	Men and women age 4–34 yr
Tanaka et al. (101)	HR_{max} = 208 − (0.7 × age)	Healthy men and women
Gellish et al. (38)	HR_{max} = 207 − (0.7 × age)	Men and women participants in an adult fitness program with broad range of age and fitness levels
Gulati et al. (47)	HR_{max} = 206 − (0.88 × age)	Asymptomatic middle-aged women referred for stress testing

HR_{max}, maximal heart rate.

Figure 3. This chart shows the various accepted forms to calculate estimated maximal heart rate according to the ACSM guidelines.

Results

Subject 1: male, untrained, 23 yrs. old, 178 cm height, 91.63 kg weight, 70% HR_{max} at 137.9 bpm

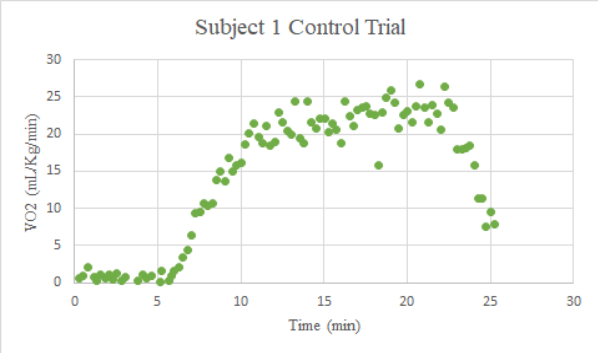

Figure 4. Subject 1 control trial without the addition of concentrated O_2. VO_2 levels begin to raise at 5.6 mins. Steady-state is reached at 22.02 mL/Kg/min at 12.3 mins. A 6.63 min O_2 deficit.

TIME	RPE	RER	HR	VO_2 relative	VO_2 absolute
Rest	0	0.61	72	0.8	0.07
Warm-up	0.5	0.67	104	9.5	0.87
2	2	0.85	120	15.8	1.45
4	3	0.91	122	20.0	1.84
6	3	0.91	124	21.7	2.00
8	3	0.89	140	20.9	1.92
10	3	0.90	146	23.6	2.17
12	4	0.93	148	22.4	2.06
14	3	0.91	136	22.8	2.09
15	3	0.89	150	25.4	2.33
Cool-down	0.5	0.97	140	11.3	1.04

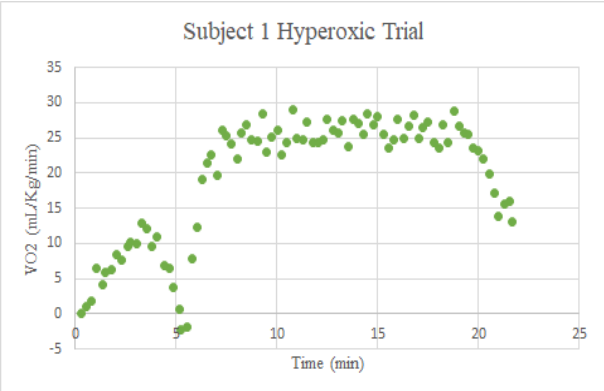

Figure 5. Subject 1 hyperoxic trial with the addition of concentrated O_2. VO_2 levels begin to raise at 0.8 mins. Steady-state is reached at 25.78 mL/Kg/min at 7.3 mins. A 6.49 min O_2 deficit.

TIME	RPE	RER	HR	VO_2 relative	VO_2 absolute
Rest	0	0.78	82	6.4	0.58
Warm-up	0	0.71	87	12.1	1.11
2	0.5	0.74	80	19.1	1.16
4	2	0.87	160	25.6	2.35
6	3	0.93	148	22.7	2.08
8	3	0.91	121	24.8	2.27
10	3	0.95	150	25.5	2.34
12	3	0.92	154	25.0	2.3
14	3	0.91	156	27.0	2.47
15	3	0.93	147	25.6	2.35
Cool-down	2	0.93	95	15.6	1.44

Subject 2: male, untrained, 26 yrs. old, 175 cm 2height, 77.11 kg weight, 70% HR_{max} at 135.8 bpm

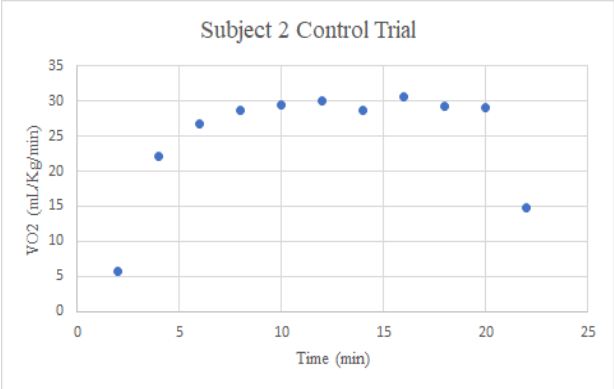

Figure 6. Subject 2 control trial without the addition of concentrated O_2. VO_2 levels begin to raise at 2 mins. Steady-state is reached 29.37 mL/Kg/min at 8.0 mins. A 6.0 min O_2 deficit.

TIME	RPE	RER	HR	VO_2 relative	VO_2 absolute
Rest	0	1.10	70	5.7	0.62
Warm-up	2	0.97	123	22.2	1.79
2	3	1.01	128	26.7	2.01
4	3	1.04	138	28.7	2.22
6	3	1.03	138	29.5	2.28
8	3	1.03	140	30.1	2.33
10	3	1.05	142	28.6	2.32
12	3	1.06	146	30.5	2.35
14	3	1.06	143	29.2	2.18
15	3	1.09	143	29.0	2.24
Cool-down	1	1.12	113	14.8	1.15

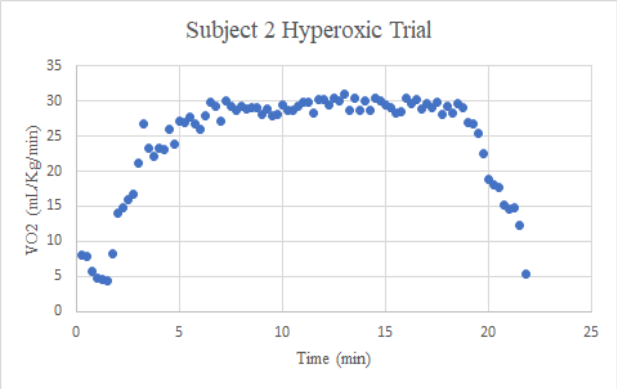

Figure 7. Subject 2 hyperoxic trial with the addition of concentrated O_2. VO_2 levels begin to raise at 1.8 mins. Steady-state is reached 29.23 mL/Kg/min at 6.3 mins. A 4.48 min O_2 deficit.

TIME	RPE	RER	HR	VO_2 relative	VO_2 absolute
Rest	0	0.94	80	4.9	0.38
Warm-up	0	1.06	89	9.2	0.71
2	2	0.99	132	27.3	2.11
4	3	0.94	126	28.2	2.18
6	3	0.98	136	29.0	2.17
8	3	0.99	136	29.9	2.31
10	3	0.97	140	30.0	2.32
12	3	0.95	144	30.4	2.35
14	3	0.96	146	30.2	2.33
15	3	0.95	142	32.1	2.48
Cool-down	2	1.02	105	16.2	1.25

<u>Subject 3</u>: male, untrained, 20 yrs. old, 168 cm height, 63.50 kg weight, 70% HR_{max} 140.0 bpm

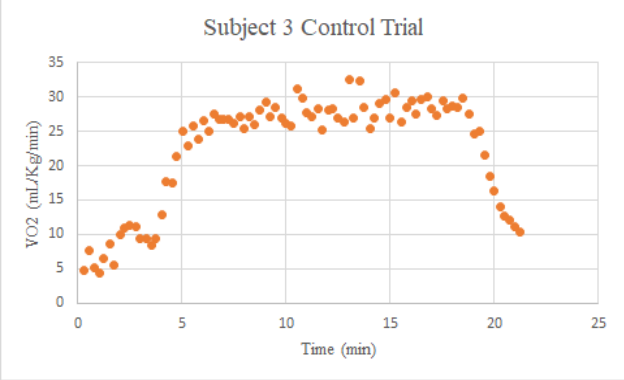

Figure 8. Subject 3 control trial without the addition of concentrated O_2. VO_2 levels begin to raise at 0.3 mins. Steady-state is reached 27.96 mL/Kg/min at 6.5 mins. A 6.25 min O_2 deficit.

TIME	RPE	RER	HR	VO_2 relative	VO_2 absolute
Rest	0	0.85	84	9.5	0.60
Warm-up	1	0.88	128	23.9	1.52
2	2	0.99	109	25.4	1.62
4	3	0.95	139	25.7	1.64
6	3	0.97	135	25.5	1.61
8	3	0.92	140	26.9	1.71
10	3	1.00	140	28.4	1.81
12	3	0.98	147	27.5	1.75
14	3	0.94	132	28.5	1.81
15	3	0.97	93	27.5	1.75
Cool-down	1	0.97	117	12.3	0.70

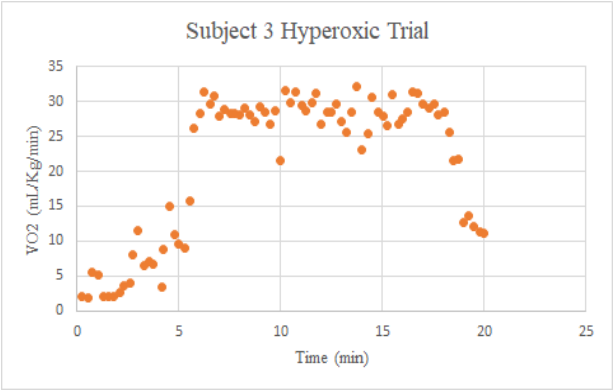

Figure 9. Subject 3 hyperoxic trial with the addition of concentrated O_2. VO_2 levels begin to raise at 0.3 mins. Steady-state is reached 28.62 mL/Kg/min at 6.0 mins. A 5.77 min O_2 deficit.

TIME	RPE	RER	HR	VO_2 relative	VO_2 absolute
Rest	0	0.85	80	6.5	0.04
Warm-up	1	0.86	94	6.5	0.13
2	2	0.79	123	6.6	0.42
4	3	1.01	126	28.4	1.80
6	3	0.97	140	28.3	1.80
8	3	0.96	139	26.7	1.70
10	3	0.93	157	28.6	1.82
12	3	0.95	144	32.2	2.05
14	3	0.96	147	26.8	1.71
15	3	0.97	150	29.1	1.85
Cool-down	2	1.05	122	12.2	0.77

Discussion

We know that if an individual is untrained, the O_2 deficit will be greater in comparison to a trained individual. A greater O_2 debt would result in a prolonged amount of time for the body's aerobic energy pathways to increase mitochondrial production to use the inspired oxygen to create ATP from ADP needed for the exercise and reach steady-

state VO_2. It was hypothesized that the inhalation of concentrated oxygen during early stages of aerobic exercise would decrease oxygen deficit, improve VO_2 kinetics, and result in quicker recovery rates. From the results of our experiment, the addition of Boost Oxygen's supplemental 95% pure O_2 at the beginning of aerobic workout did in fact result in faster VO_2 kinetics in all the three untrained male subjects. The average decrease in time in VO_2 kinetics for the three subjects was 43.2±42.7 seconds.

During the control trial for subject 1, the subject reached their average steady-state VO_2 at 22.0±2.2 mL/Kg/min, 6.63 mins after the initial raise. During the second trial the subjects achieved steady-state at 25.8±1.8 mL/Kg/min, in 6.49 mins. When comparing the first subject's control trial (fig. 4) to their second hyperoxic trial (fig. 5), the subject reduced the time to reach steady-state VO_2 by 9.6 seconds. This resulted in a 2.1% decrease in time to reach steady-state. In the second subject's control trial (fig. 6), it can be seen that the VO_2 begins to raise around 2 mins and reaches steady-state around 8 minutes resulting in roughly 6 minutes to reach steady-state at 29.0±1.2 mL/Kg/min. In comparison to the second subject's hyperoxic trial (fig. 7), the subject reached the desired steady-state at 29.25±0.8 mL/Kg/min at *one and a half minutes faster* at 4.48 mins, from the initial VO_2 kinetics. Resulting in a 25.3% faster than in the control trial. Subject 3 reached steady-state 7.9% faster, 28.8 seconds, in comparison to the control trial.

A possible positive finding from the results of the study show that supplemental concentrated O_2 can help create a more stable steady-state VO_2. When comparing the control trial to the hyperoxic trial of subjects 1 and 2, there is a more consistent steady-state VO_2. The first subject decreased the standard deviation of the average steady-state VO_2 by 0.46. The second subject decreased the standard deviation of the average steady-state VO_2 by 0.34. A few outlier points caused the third subject's hyperoxic trial to vary more in steady-state VO_2 in contrast to the control. The third subject's average steady-state VO_2 increased 0.39. This increased stability of steady-state VO_2 in two of the three subjects was not an anticipated finding and could potentially have positive effects on aerobic performance.

Although it was hypothesized that the data would show faster recovery rates due to the increased oxygen during post-exercise cool-down and shortly after, contrarily, the results did not indicate any significant increase or decrease in recovery rates. It is plausible that the additional supplemental oxygen that was inhaled at the initial stages of exercise was metabolized during the exercise trial. During the exercise, the subjects reported an average RPE score of 3, or moderate perceived exertion. There was no change to RPE scores with the addition of Boost Oxygen's supplemental oxygen.

There are some factors from the experiment that may or may not influence the results. One factor that was known to the researchers was the presence of two small holes located on each side of the mouthpiece spouts of the Boost Oxygen 2-liter canisters. The mouthpiece spout is where the oxygen is transferred from the canister to the person. These holes allow atmospheric air to be inhaled in addition to the 95% pure oxygen from the canister. With the atmospheric oxygen being around 20% of atmospheric air, it can only be certain that the subjects were inhaling somewhere between 20-95% oxygen with each inhalation of the canister. In the hyperoxic trials (fig. 5, 7, and 9), during the initial rise of VO_2 there is an increased variance in the scatter plot while reaching steady state in contrast to the

control trials. The variance in these graphs were caused by the transfer from the spirometer mouthpiece, to the mouthpiece spout of the O_2 canister and back to the spirometer mouthpiece. Furthermore, during each of these transfers it is expected that a very minimal amount of atmospheric air enters the mouthpiece of the spirometer. The atmospheric air is not constantly recorded while removed from the subject, only the air that is in the small inner volume of the mouthpiece is added to the recording of the exhales. Although minimal, it may have a small effect on the data. Ideally, the concentrated oxygen would be administered directly into the sealed spirometer mask to prevent the small amount of atmospheric air to enter. This could also prevent some of the atmospheric air from entering from the two holes in the mouthpiece spout, thus increasing the O_2 concentration in each inhalation.

It is acknowledged that the necessity of a warm-up can be argued during this study. An extensive warm-up can negatively skew the data causing a prolonged time to reach steady-state. We decided to include a very mild warm-up to allow the subjects to prepare their bodies for the upcoming exercise trial. It can be noted in the charts of the results that during the trials, the 70% HR_{max} was not perfectly maintained. The treadmill speed was adjusted during the workout to come as close to the target HR as possible.

Due to a small subject group, there can be many improvements to the study. A larger cohort group with the expansion of the following factors; age, gender, aerobic training status, weight, fat percentage, smokers/non-smokers, residence location (for altitude acclimation), and genetics/race would be most ideal to further understand the effects of supplemental concentrated oxygen in aerobic exercise. The trials in this study were not randomized, therefore a learning curve would be eliminated if the control and independent trials were in alternating or randomized order.

Conclusion

From our study's findings, we can assume that Boost Oxygen's supplemental oxygen canister can cause improvements in VO_2 kinetics and reduce oxygen deficit when administered just prior to aerobic steady-state exercise in untrained individuals. Furthermore, supplemental concentrated oxygen may help maintain a steadier steady-state VO_2.

Our hypothesized acceleration in recovery rates were revealed to be invalid from the results. There was no significant increase or decrease in recovery rate when administering oxygen at the beginning of an aerobic exercise. However, although there were no significant changes in recovery in our study, taking oxygen post-exercise may produce different results. The findings of this cohort study can prove to be beneficial to any athletes, trainers or active individuals who aim to shorten the time it takes to reach steady-state VO_2.

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